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ORIGINAL COMMUNICATION

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Vestibular neuritis: Vertigo and the high-acceleration vestibulo-ocular reflex

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Abstract Patients after vestibular neuritis (VN) often report persistent dizziness and disequilibrium. We correlated persistent symptoms with sustained impairment of the high-acceleration horizontal vestibulo-ocular reflex as determined by quantitative search-coil head-impulse testing (qHIT). In 47 patients, qHIT was recorded 0–60 months and symptoms assessed with the Yardley Vertigo Symptom Scale short form ≥18 months after VN onset. No correlation between the magnitude of high-acceleration vestibular impairment and the severity of vertigo symptoms was observed. The lack of a symptom-qHIT correlation suggests that defective compensation at a more rostral level in the central nervous system may be responsible for protracted symptoms in VN patients.

Key words head-impulse test · questionnaire · neuro-otology

Introduction

Vestibular neuritis (VN) is defined as a sudden unilateral deficit of the peripheral vestibular organ (labyrinth or nerve) without auditory symptoms in otherwise healthy subjects. Signs and symptoms result from an imbalance of the tonic discharge between the impaired and intact vestibular afferents. Recovery is due to a combination of peripheral vestibular restoration and central compensation [4]. The time course of recovery varies between individuals and, contrary to conventional clinical knowledge [3], almost 50 % of patients with VN report sustained dizziness and disequilibrium [2, 5, 6, 9, 11, 12]. Vestibular function when assessed by caloric irrigation, rotatory chair testing, posturography, or clinical balance testing does not seem to differ between patients with sustained symptoms and symptom-free patients [2, 6, 10–12]. However, a limitation of the latter studies is that they all rely on measurements of low-frequency components of vestibular function. This is important because Schmid-Priscoveanu et al. showed that the low-frequency vestibular function, as determined by caloric irrigation, becomes symmetrical, whereas the high-frequency function, assessed by quantitative search-coil head-impulse testing (qHIT), often remains impaired [14]. Since the vestibular apparatus is most efficient when transducing high-frequency components of head motion, such as occur during locomotion, it would be reasonable to expect symptoms to correlate with the qHIT. We therefore set out to compare sustained residual vestibular symptoms, as assessed by the shortened version of the Yardley Vertigo Symptom Scale (sVSS), with qHIT in VN patients.

Methods

Subjects

Forty-seven patients (27 male, 33 – 87 y [49 ± 15]) diagnosed with unilateral peripheral vestibular hypofunction due to VN participated in the study. All patients had developed acute vertigo without additional auditory or neurological symptoms and displayed spontaneous horizontal-torsional nystagmus and a pathological unilateral bedside head-impulse test during clinical examination. Patients were divided into two groups depending on the time interval between VN onset and the date of qHIT (acute: 1 day – 4 weeks, N = 14; chronic: > 4 weeks – 60 months, N = 33). The acute/chronic cutoff point was based on a previous study showing that the vast majority of improvement in qHIT takes place within 4 weeks after VN onset [13]. Twelve acute patients were tested twice at different times after VN onset; the first qHIT measurement was within the first 4 weeks after VN onset in all 12 patients and the subsequent measurement was after 4 weeks (first qHIT: 1.3 SD 1.5 weeks; subsequent: 13 SD 19 months). The comparison group for qHIT data comprised 28 healthy subjects (13 male, 18–75 y). Informed consent was obtained from all participants and the protocol was approved by the local ethics committee, in accordance with the 1964 Declaration of Helsinki.

quantitative head-impulse test (qHIT)

Three-dimensional eye and head movements were recorded in a magnetic frame (Remmel type system, modified by A. Lasker, Baltimore, MA, USA) using dual search-coils. The search-coils were calibrated before each session (see Straumann et al. [15] for details). Horizontal head impulses (amplitude: 20–40°; duration: 150–200 ms; peak velocity: ~300 °/s; peak acceleration: ~10000 °/s^2) were applied by an investigator standing behind the subject who was visually fixing upon a target light 1.24 m straight ahead. The direction of the head impulses was pseudorandomized and four to six impulses were performed in each direction. The gain of the horizontal vestibulo-ocular reflex (VOR) was determined by computing the coefficient 'eye-in-space displacement divided by head-in-space displacement' as head-in-space moved from 3° to 7° eccentricity from straight-ahead. We used this analysis to minimize any possible effect of latency on gain, since it is not known whether VN affects only the gain or, in addition, also the latency of the VOR. An increased latency, however, would lead to a false reduction of gain if determined at peak head velocity [13].

Gain asymmetry ($gVOR_{asym}$) in percent was computed by:

$$gVOR_{asym} = \frac{gVOR_{contra} - gVOR_{ipsi}}{gVOR_{contra} + gVOR_{ipsi}}$$

whereby $gVOR_{ipsi}$ denotes the VOR gain during head impulses to the ipsilesional side and $gVOR_{contra}$ the VOR gain during head impulses to the other side.

Vertigo symptom scale

Two to 98 months (46 SD 27) after vestibular examination, patients were asked to complete the shortened version of the Yardley Vertigo Symptom Scale (sVSS), measuring the frequency of dizziness, vertigo, imbalance, and related autonomic symptoms during the past 12 months [19–21].

Results

Fig. 1 plots gains of the horizontal VOR for qHIT towards the ipsilesional side (Fig. 1, left) and gain asymmetry between the contra- and ipsilesional side (Fig. 1, right) as a

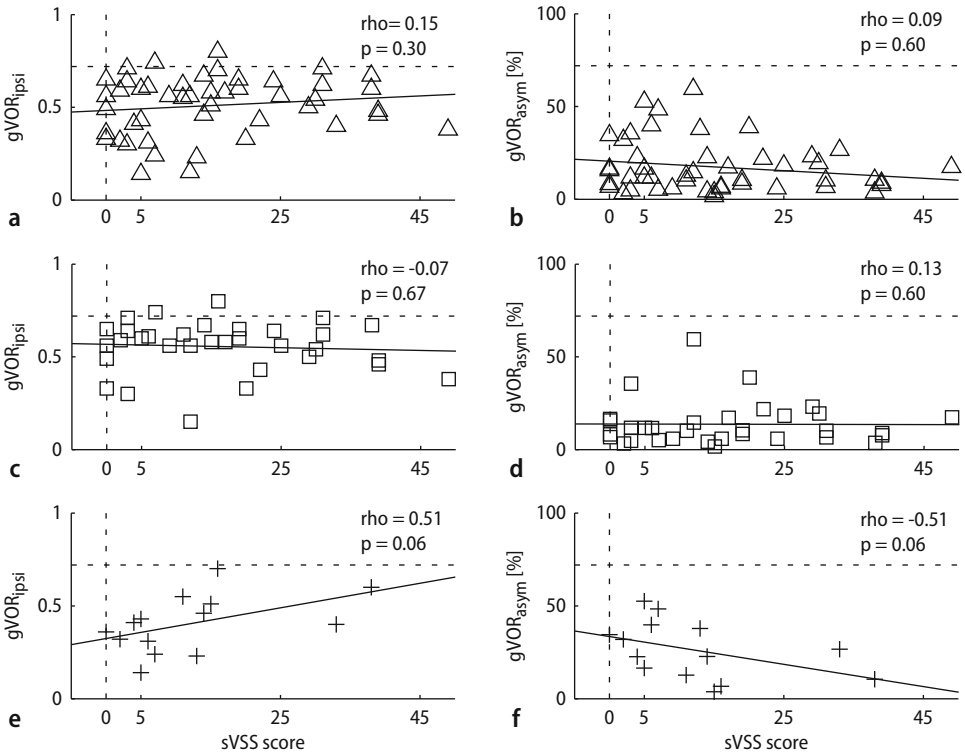

Fig. 1 Horizontal VOR gains during qHIT towards the ipsilesional side (left diagrams) and gain asymmetry (right diagrams) plotted as a function of sVSS scores. Triangles (**a, b**): all patients (N = 47); squares (**c, d**): chronic patients (N = 33); crosses (**e, f**): acute patients (N = 14). $gVOR_{ipsi}$ VOR gains for ipsilesional qHIT; $gVOR_{asym}$ percentage of gain asymmetry between contra- and ipsilesional VOR gains (see Methods for definition). Horizontal dashed lines: lower limit of average VOR gains for healthy subjects – 2SD; vertical dashed lines: zero sVSS score corresponding to complete symptom absence. Diagonal solid lines: linear regression slopes optimized by linear squares estimation; the corresponding statistics (rho and p) indicate the strength of the relationship between the two variables obtained with nonparametric Spearman statistics. Post hoc statistical power analyses for linear regressions with one predictor revealed adequate samples sizes for acute and chronic patient groups (power > 0.80)

function of sVSS in all patients (Fig. 1 A, B) as well as in the chronic (Fig. 1 C, D) and acute (Fig. 1 E, F) groups. Age was not significantly different between symptom-free and symptomatic patients (unpaired t-test: p = 0.6). Neither in all patients (Fig. 1 A, B) nor in the chronic patient group (Fig. 1 C, D) did ipsilesional qHIT gains or gain asymmetries correlate with sVSS scores (Spearman rank correlation coefficients ranging between –0.07 to 0.15, p-values > 0.1). In acute patients, contrary to what may be expected, low ipsilesional qHIT gains (Fig. 1 E) and large gain asymmetries (Fig. 1 F) tended to relate to low sVSS scores (i.e., less vertigo), but the correlation was not significant (gVORipsi: Spearman rank correlation = 0.51, p = 0.06; gVORasym: Spearman rank correlation = –0.51, p = 0.06).

Fig. 2 shows ipsilesional qHIT gains (A) and corresponding gain differences between the two assessments (B) plotted as a function of sVSS scores in the 12 patients who were tested twice after VN onset. The interval between the first (Fig. 2 A, circle) and the subsequent (Fig. 2 A, cross) measurement ranged between 1 and 60 months (13 SD 19 months). Low sVSS scores appeared to correlate with large ipsilesional gain improvement (Fig. 2 B), although this correlation was not significant (Spearman rank correlation = –0.3, p = 0.3).

Discussion

Our study demonstrates that persistent dizziness after VN is not significantly associated with sustained vestibular impairment as assessed by the quantitative search-coil head impulse testing (qHIT). More specifically, severe vestibular deficit in the chronic patient group did not imply a high score on the shortened version of the Vertigo Symptom Scale (sVSS), assessing dizziness, vertigo and imbalance during the past 12 months. Although a lack of congruency between persistent symptoms and vestibular function tests has been described previously in patients with VN [2, 5, 10–12], this is the first study showing that this conclusion also holds for the high-frequency components of vestibular function.

The fact that sVSS assessment in our study was conducted on average about 4 years after vestibular examination could be viewed as a limitation of this study. However, our explicit aim was to ascertain the presence of protracted symptoms in VN patients. We do not expect that this time interval had a major impact on the correlation of vestibular function in chronic VN patients, since qHIT measurements were also obtained when peripheral recovery and/or central compensation were stabilized [13]. Furthermore, the lack of correlation between persistent symptoms and high-frequency VOR function is unlikely to depend on sample size, as revealed by post hoc statistical power analysis (see legend Fig. 1).

A potentially interesting observation was that the two patients with very high sVSS scores showed a combination of only mild to moderate ipsilesional gain impairment and scarce gain improvement over time (Fig. 2). This finding, which might suggest a greater risk to develop chronic vertigo if gain impairment or improvement are less pronounced, needs to be verified in more patients. Such an analysis is particularly important in the light of recent suggestions that steroid treatment for acute VN improves peripheral-vestibular recovery (albeit measured with the caloric test and without symptom assessment) [1, 16].

What could be the main determinants of the long-term clinical recovery after acute VN given that residual symptoms are not correlated to conventional vestibular function tests (i.e., caloric or qHIT)? First, persistent otolith imbalance could account for sustained vestibular symptoms. To our knowledge, the only study assessing utricular function (typically affected in VN) and vestibular symptoms did not find differences in static ocular counterroll and subjective visual vertical responses

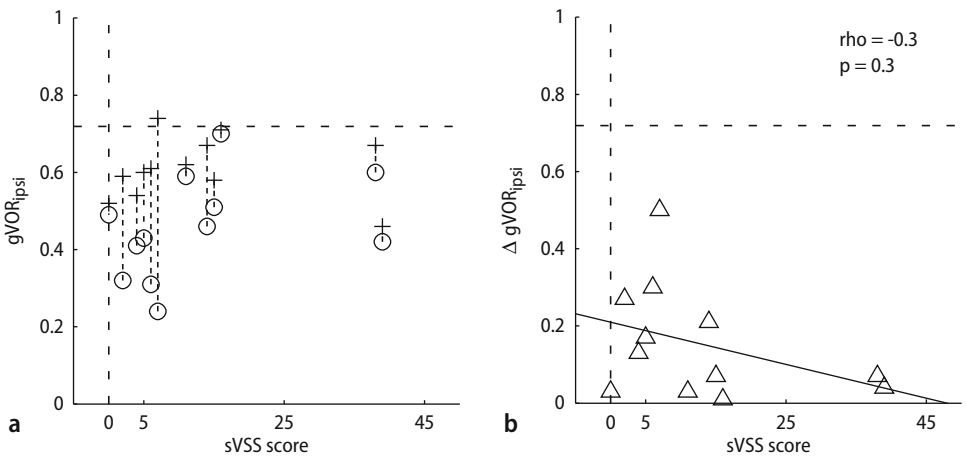

Fig. 2 Horizontal VOR gains for the first and subsequent ipsilesional qHIT measurement (**a**) and corresponding gain differences (**b**) plotted as a function of sVSS scores in patients tested twice after VN onset (N = 12). Circles: ipsilesional VOR gains (gVOR$_{ipsi}$) resulting from first qHIT measurement (i.e., within 4 weeks after VN onset); crosses: ipsilesional VOR gains (gVOR$_{ipsi}$) resulting from subsequent qHIT measurement (i.e., > 4 weeks from VN onset). $\Delta\ gVOR_{ipsi}$ = gVOR$_{ipsi}$ first measurement – gVOR$_{ipsi}$ subsequent measurement. Solid and dashed lines as in Fig. 1

between symptom-free and symptomatic VN patients [22]. Second, enhancement of the deficient VOR by early catch-up saccades, a strategy which assists gaze stabilization [18], but is not routinely assessed, may be insufficient in some patients. Recently, it has been shown that patients with a persistent abnormal bedside head-impulse test were more likely to be dizzy [22]. Possibly, in these patients, catch-up saccades do not occur during (covert saccades), but after the head impulse (overt saccades), which is easier to detect at the bedside [23]. Finally, deficient cortical adaptation, including psychophysical and psychological processes, could be responsible for protracted symptoms in VN patients [6–8, 17]. Longitudinal assessments of such central processes in VN patients, however, remain the purpose of further investigations.

Disclosure

The authors have reported no conflicts of interest.

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